Modern Concepts of Cardiovascular Disease

Published monthly by the AMERICAN HEART ASSOCIATION

50 WEST 50TH STREET, NEW YORK, N. Y.

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Vol. VI October, 1937 No. 10

THE MECHANISM OF THE PRODUCTION OF HEART MURMURS

PART II

The chief factors modifying audibility of murmurs are these:

(1) *The direction of blood flow.* Murmurs, in the main, are transmitted best in the direction of the current. This depends upon the fact that the eddies are propagated and released in this direction, but more through the fact that the point of impact of the stream upon a solid body must be distal to the obstruction. However, the murmur, if loud enough, will be transmitted somewhat in all directions by local resonators and direct continuity of solid bodies.

(2) *The size of the ostium,* in the line of either forward or backward flow, in relation to the velocity. Too small a stenosis with its resulting frictional effect upon velocity may give less murmur if the pressure gradient be reduced by cardiac failure, than a larger orifice. Conversely, a larger ostium may pass blood more readily at lower velocity with less turbulence and softer murmur than a smaller orifice. In other words, pressure behind the obstruction must always be considered in relation to the caliber of the individual channel and to the diameter and pressure beyond the obstruction.

(3) *Valve forms.* Obstructions on the up-stream side of a narrowed channel may cause murmurs in flowing fluids. Here, however, sharp edges are more provocative of turbulence and relatively higher velocities are necessary for audibility. The anatomy of injured valves, however, comes into question here. Apparently a cone-shaped or straight-edged orifice down-stream will produce a loud murmur, whereas a funnel-shaped outlet damps the vibrations. A cone with its apex and inlet pointing up-stream produces very soft murmurs even at high velocities. Bondi has used this experimental finding in explaining the extreme softness or absence of a diastolic murmur of aortic regurgitation in many cases of aortic stenosis, showing that in the diastolic phase the damping effect of the cone-shaped valves directed against the current is further augmented by the damping effect of the funnel-shaped opening into the left ventricle from the aorta.

(4) *Local resonance and damping of sound.* The material of which fluid conductors are made determines the audibility of sound vibrations originating in them. A practical instance of this in engineering is the fact that water pipe noises have progressively increased as domestic plumbing has changed from iron, to brass and finally to copper pipe. It seems likely that the physical condition of heart muscle, including the papillary muscles, the valves and the great vessels, has some modifying influence upon sound transmission. Certainly the apposition of the region of impact of the turbulent stream to the chest wall is of considerable importance. The overlying lung is a poor conductor. The energy loss of vibrations in passing from a solid body to air may be as much as 99%. Moreover, the sound vibrations are readily damped by the blood away from the point of impact against the walls of heart or blood vessel. Because of this, the entrance of a jet into a chamber of high pressure causes less murmur. As an example, a mitral regurgitant murmur in mitral stenosis may be damped when the patient is in severe failure with a high pressure in the left auricle, or indeed it may explain in part the absence of demonstrable mitral regurgitation in cases of "pure" mitral stenosis. Heart size, shape and position all act as modifying factors, varying the contact with the chest wall. Angulation of the great vessels may be of some importance. Cossio points out the increased torsion of the aorta in patients with transverse hearts in whom aortic systolic murmurs are often found, and a comparable torsion of the pulmonary artery with the common appearance of a murmur in the vertical heart.

While local resonators, such as bone, and influences damping sound, vibration such as thick chest wall, vary greatly with different pathological and anatomical conditions, the whole acoustical problem of the chest is complicated by the reflection and refraction of sound and the final appearance of a murmur at the chest wall is through a tortuous course where different factors have influenced its intensity, pitch and quality. The working of such influences makes it understandable why, in a series of normal individuals, the intensity of the loudest first heart sound recorded by Williams and Dodge (1926) had forty times the energy of the weakest.

(5) One other factor is the *viscosity of the blood.* Of how much practical import a change in viscosity may be in producing heart murmurs is difficult to say. In anemia, other mechanisms increasing blood velocity come into play, such as increased stroke volume and lowered peripheral resistance as well as cardiac dilatation. Judging from experiments, a change from water to a glycerine-water mixture with the viscosity of blood requires an increase of velocity of about 20-30 cm. per second for the production of a minimal murmur, and this lowering of the critical velocity with the less viscous blood is probably of some moment in revealing small valvular defects or valvular incompetence with ventricular dilatation in cases of high grade anemia.

(6) *Variation of stethoscope form* by emphasizing different frequencies will alter the audibility of minimal murmurs very considerably. The diaphragm or Bowles type is more critical for high, and the bell type for low, frequencies (Sprague, 1926).

Murmurs not due to valvular or septal defects. The term "functional" murmur implies a murmur whose mechanism of production is not dependent upon deformity of the valves or the cardiac septa. It usually includes murmurs resulting from disturbance of the valvular action, as in dilatation of the heart chambers or valve rings, as well as murmurs produced outside the heart. To understand the basis for some of these murmurs, one must appreciate certain features of valvular closure. It has been shown by several observers, as far back

as Baumgarten in 1843, that the valves may close without a leak, which must presuppose some action effective in their closure beyond the simple hydrostatic back pressure at the time that it becomes greater in the distal chamber and overbalances that in the proximal. If that mechanism alone closed the aortic valve, for example, the regurgitation of blood into the left ventricle would approximate ten per cent of the stroke volume (Hochrein). Henderson and Johnson in 1912, following the earlier idea of Ceradini, demonstrated that the mitral valve is practically entirely closed at the end of ventricular diastole and that, at slow heart rates, this closure is accomplished by the sudden breaking of the jet of blood entering the left ventricle from the auricle. This jet moving into the blood in the ventricle at the end of auricular systole draws behind it a region of negative pressure like the wake of a ship which abruptly sucks together the free margins of the cusps. Where no such final jet occurs due to the absence of auricular systole, as in auricular fibrillation, at slow rates when active diastolic filling is much reduced in presystole, the valves close with a hinge-like action and regurgitation must take place when the closure is accomplished merely by the static pressure changes. In a similar fashion, Hochrein has shown that the aortic valves normally move toward each other during ventricular discharge and are so close at the end of systole that the normal regurgitation is very slight. At the pulmonary valve in man, the normal regurgitation has been calculated at only 0.51 — 1.47 c.cm. This inward bowing of the aortic and pulmonic valves results in eddy formation in the sinuses of Valsava which assist the complete closure of the valves at the beginning of diastole. Bondi has suggested that the reason aortic systolic murmurs are not heard normally in every person may be dependent upon this nozzle or injector action at the valve, whereby the stream tends to be delivered less abruptly and more directly into the center of the aorta and thus its vibrations are damped by a thicker layer of blood about the central jet. However, other mechanisms of the normally contracting ventricle which result in lowered velocity at the aortic ring are probably effective.

It seems reasonable to invoke the disturbance of these remarkable adaptive mechanisms in cardiac failure in explaining some mitral and aortic regurgitant murmurs when inadequate discharge through the valve in the previous phase may have been ineffective in bringing the valves into an ideal position for abrupt closure.

Dilatation of the ventricles has been considered a cause of some functional murmurs — especially of the mitral systolic and apical diastolic (Austin Flint) type. So far as the mitral systolic is concerned, it has been thought dependent upon a stretching of the valve ring with imperfect closure of the valves. Another explanation is that the enlargement of the ventricle is not associated with a comparable elongation of the papillary muscles and they, therefore, hold the valve partly open. These considerations are theoretical and in all intracardiac functional murmurs one must postulate that the turbulence within the ventricles is probably modified not only in production, but in transmission by alterations in muscle tone. The Austin Flint murmur has been called the murmur of relative mitral stenosis, either with the assumption that the regurgitant stream from the aortic valve presses upon the aortic leaflet of the mitral valve to obstruct the flow of blood into the ventricle, or that the dilatation of the left ventricular cavity seen with aortic regurgitation makes the normal mitral valve relatively small. The finding of Herrmann in two cases with loud Austin Flint murmurs that the posterior cusp was the one involved, supports the previous view, while that of White and Wood (1923), and Bland, White, and Jones (1935) that the murmur may occur with ventricular dilatation in the absence of aortic regurgitation, is in favor of the latter. A third suggestion, that the murmur represents an impact between the forward stream from the auricle and the backward stream from the aorta can still be considered plausible.

The murmur of pulmonary regurgitation (Graham Steell) is found with pulmonic hypertension and has been thought of as being a blowing back through the pulmonary valve of a high pressure stream from the pulmonary artery through a valve rendered incompetent by stretching of the cusps or the valve ring. One may speculate that failure of the weakened right ventricle to discharge a proper jet into the pulmonary artery against the high pressure may interfere with the mechanism for the physiologic apposition of the valves early in diastole, as has been mentioned in the discussion of aortic regurgitation.

To what extent normal or abnormal structures in the heart chambers, especially the ventricles, can cause the formation of audible eddies is unknown. Bands of muscle, bacterial vegetations, anomalous chordae and papillary muscles, have been held responsible. Further correlation between auscultatory findings and anatomy is needed.

Murmurs heard over the heart may be extracardiac in origin. Ortiz (1933) finds that friction between serous surfaces of the heart and pleurae may be important. The *cardiorespiratory murmur* is apparently produced by the movement of air in the alveoli of the lungs in contact with the heart. Systolic contraction causes a negative pressure in the region of the heart and air is drawn into the overlying lung. Diastolic filling may displace this. The more abrupt change of volume with systole explains the systolic timing of cardiorespiratory murmurs.

Some distant humming murmurs, either systolic or diastolic, arise in the vessels of the mediastinum. They are probably largely venous in origin. Other vascular murmurs at times heard in the chest are those of the normal venous hum above the clavicles accentuated by constriction of the veins by extending and rotating the head; the murmur of aneurysms; and the murmurs of patent ductus arteriosus and coarctation of the aorta. All of these are explicable in terms of the entrance of a jet of blood into a vessel of wider caliber at sufficient velocity.

Summary

Cardiac murmurs are audible vibrations produced in the circulating blood by the formation of rhythmic eddies. Such eddies are the results of the disturbance of streamline flow produced where regions of discontinuity occur when streams of different velocity are apposed. This takes place mainly where the change of velocity occurs as a relatively narrow jet of fluid is projected into a vessel or chamber of wider diameter. To be audible the jet must have a velocity above a critical level. The velocity is determined not only by the diameter of the stream but by the pressure difference behind and ahead of the constriction. Local conditions of resonance and damping markedly affect the audibility of murmurs. Both valvular anatomy and valvular function are factors in the production of murmurs. Ventricular dilatation and cardiac failure may produce, alter, or suppress heart murmurs. Unexplained and probably normal turbulence in the flow of blood in the heart may account for some accidental murmurs. Many others are produced outside of the heart in serous surfaces or in the lungs.

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